on a quick answer for the cause of death, thus not allowing adequate investigation in cases of this nature? Why does the coroner have to pronounce his verdict at all in these cases, when there exists a panel of experts more qualified to decide—that is, the P.M.P. or the U.I.C.C. Consultative Committee?

Dr. Thurston points out that "recording of causes of death is important for the national statistics on which so many calculations and decisions depend." The asbestos industry is fully aware of the health hazards associated with it, but inaccurate death certification can only serve to give the wrong impression and harm an enlightened industry producing a product for which in many instances there is no known substitute.—I am, etc.,

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Avoidable Hazards

SIR,—Your leading article "Asbestosis" (8 July, p. 62) was most timely. It is probably not realized that asbestos is used by some dental surgeons in postoperative packing materials following the operation of gingivectomy. It is very likely that a considerable number of dental nurses are exposed to quite heavy concentrations of powdered asbestos in the preparation of these dressings. I have noticed that some nurses shake the bottle before opening. A white cloud of fine asbestos powder appears immediately the lid is removed.

As there are other dressing materials available which do not contain asbestos and which are just as good there is no justification in continuing to use these preparations.—I am, etc.,

Leeds 16. JOHN E. DEB. NORMAN.

Chloasma and the "Pill"

SIR,—Dr. D. B. E. Quamina observed (3 June, p. 638) that chloasma is becoming more common as a side-effect of oral contraceptives, especially in Caucasians living in sunny countries. He says that very little reference has been made to this contraceptive chloasma and believes that analysis of a large series may help to determine whether it is the oestrogen or the progestogen in the pill which causes this complication. This presupposes that only one of these hormones is culpable. Oestrogen alone can cause chloasma, even in the male,[1] but I have not come across chloasma caused by progestogen in the absence of oestrogen. It is my opinion that the progestogen acts on the oestrogen-primed melanocytes,[2] and that chloasma is most likely to be precipitated by sunlight in chloasma-prone patients who have been taking anovulants for a long time, or in high dosage. Thus marked chloasma has been reported from Europe after as little as three months' medication with the large doses of anovulants sometimes used to control gynaecological troubles[3]; and several surveys have shown that the incidence of chloasma increases with the length of time for which the anovulant has been taken.

From various series already published it can be concluded that, for a given oestrogen dosage in a "combined pill," the greater the dosage of progestogen the greater the incidence of chloasma. Changing from one brand of anovulant to another would thus be of no help unless there was a reduction of hormone dosage. Chloasma can also be caused by "sequential therapy."[4]

Dr. Quamina, in Trinidad, is in an excellent position to help to unravel the chloasma mystery. The incidence of chloasma among the anovulant-taking women of another Caribbean island (Puerto Rico) is reported to be as high as 34%.[4] Is there a similar percentage among the natives of Trinidad? I had imagined that the most deeply pigmented races would be chloasma-free, but it has been stated[5] that chloasma can be seen frequently in the African negro woman if a careful examination is made.—I am, etc.,

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Antinuclear Factors

SIR,—The comment in your leading article on "Antinuclear Factors" (10 June, p. 654) that "despite the absence of any certain evidence it may be safely concluded that they have some role in the pathogenesis of [connective tissue] disease" is in our view incorrect.

Only 84% of patients suffering from systemic lupus erythematosus, 81% of patients with systemic sclerosis, and 35% of patients with discoid lupus erythematosus have antinuclear antibodies, even in the active phase of the disease.[1] The titres of antinuclear antibodies are not necessarily proportional to the activity, progress, or treatment of disease.[2] These antibodies are found in several diseases and no antibody is characteristic of any particular disease. They are found in symptomless relatives of patients suffering from connective tissue diseases[3,4] as well as 4% of control subjects not suffering from connective tissue diseases.[1] Antinuclear antibodies cross the placenta without harming the foetus or newborn child,[5–7] and have been transfused into human volunteers without causing disease.[8–10] Transfusion of serum containing L.E. cell factor does not cause any damage to rats made tolerant to human serum proteins[11] nor does it inhibit the growth of cells in tissue culture.[12–13] Thus there is no evidence that antinuclear antibodies can initiate tissue damage.

Nevertheless, tests for antinuclear antibodies are valuable in clinical practice. In a recent assessment of the value of one test (the rat liver immunofluorescence test) we[14] have found that the connective tissue diseases can be divided into two groups. The first group includes disorders like systemic and discoid lupus erythematosus and systemic sclerosis in which high titres of antinuclear factor are frequently found, and the second those diseases like polyarteritis nodosa, various types of vasculitis, and dermatomyositis in which antinuclear factor is usually absent. We concluded that (*a*) the presence of antinuclear factor in high titre (more than 1 in 64) usually indicates a connective tissue disease such as systemic or discoid lupus erythematosus, systemic sclerosis, Sjögren's syndrome or rheumatoid arthritis, and almost certainly excludes polyarteritis nodosa or dermatomyositis; (*b*) patients with a titre of 1 in 64 or higher and showing no or few abnormal symptoms or signs should be followed for a long time because they are at risk to develop a connective tissue disease of the first group; and (*c*) in the absence of any other evidence of connective tissue disorder low titre antinuclear antibodies (1 in 16 or less) can be ignored.

The role of antinuclear antibodies in human disease has still to be determined—We are, etc.,

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Combined Antifungal and Antibiotic Therapy

SIR,—Professor P. C. Elmes's assertion in *Prescriber's Journal*[1] that "one in five strains of *Monilia* are resistant to nystatin" has been refuted effectively in the pages of the *B.M.J.* by such distinguished and authoritative writers as Professor E. Drouhet (18 March, p. 699), R. Holt and R. L. Newman, G. T. Stewart, and H. I. Winner (1 April, p. 51). We are in full agreement with these previous correspondents, having in 11 years of extensive use of nystatin encountered many therapy-resistant patients, but no resistant strains of *Candida albicans*. Permit us, however, to comment on another aspect of P. C. Elmes's article which has received rather less attention.

Professor Elmes considers that "antifungal agents are only necessary in serious infections or if local lesions persist after the tetracycline is stopped. . . . Combination tablets such as Mysteclin or Lederstatin are therefore unnecessary. . . ." Fleeting reference is made to systemic candidiasis, but the practitioner is left without guidance about the prevention of such dissemination in patients receiving prolonged antibiotic therapy. Combined anticandidal and antibacterial therapy is strongly indicated in patients in whom oral and gastrointestinal candidal reservoirs may become a focus for systemic invasion or widespread mucosal and

cutaneous involvement. This applies particularly to infants with a history of oral, cutaneous, or gastrointestinal candidiasis, as well as to patients with debilitating diseases predisposing to candidal infection (diabetes mellitus, leukaemia, etc.). Such patients frequently receive prolonged antibiotic therapy, and the combination of nystatin with tetracycline is prudent here in order to prevent systemic candidal dissemination from gastrointestinal foci. Systemic candidiasis is markedly underdiagnosed; in P. C. Elmes's own words, it is "seldom suspected . . . and . . . the diagnosis is usually made at autopsy."[1] Yet systemic candidal infections are increasing in frequency, particularly among patients with leukaemia and lymphomas. A 15-fold increase of such infections in the past decade was revealed by necropsy studies at an institution devoted to the treatment of malignant diseases.[2] We ourselves have recognized more than a score of such cases during the past few years at a 400-bed general hospital in New York City. As many as four patients at a time have been under treatment in the wards, some of them successfully.[3]

The cost tables given by Professor Elmes show that combinations of nystatin and tetracycline (Mysteclin, Lederstatin) cost only slightly more (29s. 3d.) than tetracycline capsules alone (22s. 6d.). Although the price of nystatin is not listed, the cost of administering the two antibiotics separately would probably be considerably higher.

Possible improper or promiscuous use by some practitioners of a useful combination does not warrant its removal from the pharmacopoeia. This would be as illogical as the removal of parenteral penicillin because too many injections are given. Good therapy demands the thoughtful prescription of drugs that fit the needs of a carefully investigated patient suffering from a specific type of infection. There are instances when the combination of an antibacterial and an antifungal agent is the treatment of choice, or when possible error on the side of prudence is preferable to a perfect diagnosis made post mortem.—We are, etc.,

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Protective Helmets and Traumatic Epilepsy

SIR,—Two points call for comment in the interesting article by Dr. R. H. P. Fernandez (24 June, p. 830). The case for "skidlids" for motor-cyclists has been argued on statistics which showed how seldom severe head injuries occur in victims who have been wearing a protective helmet. If Dr. Fernandez, with the large amount of data which he evidently has for industrial head injuries, could quote the incidence and mortality of injuries in men wearing helmets it might strengthen the case for compulsory protection in certain occupations. It would also be interesting to know the number of unprotected victims for whom helmets had been recommended, and perhaps even provided, by the management, and who had ignored this advice. Certainly it is an impression that helmets are much more widely worn in Continental Europe and in the U.S.A., at least in the building industry, which is seen at work by the passing tourist.

Dr. Fernandez rightly devotes space to the problem of traumatic epilepsy, which is one of the commonest causes of permanent disability—that is, non-employability—after head injury. Because this often develops in patients who have otherwise fully recovered, it is particularly important to understand its natural history in order to advise both the patient and his employer. Dr. Fernandez quotes Caviness as having shown that in many patients the epilepsy is a temporary affair with a good prospect of remission. However, this conclusion was based on a study of gunshot wounds from the first world war, and there are many differences between this population and civilians suffering blunt head injury. I have previously discussed in these columns the prediction of epilepsy after blunt injuries.[1] Elsewhere I have emphasized the less favourable outlook for remission after blunt head injury compared with missile injuries.[2] Briefly, early epilepsy (within a week of injury) has a 75% chance of permanent remission, but epilepsy developing later (whether or not there has been early epilepsy) persisted in more than 80% of 200 patients followed up for more than two years after their first fit. For the industrial medical officer the practical implications of this are that if a man does develop epilepsy he should be permanently redeployed in a job which is compatible with the occurrence of an occasional seizure, rather than offered temporary arrangements in the belief that the epilepsy will soon cease to be a problem.—I am, etc.,

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Enuresis and Psychiatry

SIR,—May I add to the points made by Dr. Ian G. Wickes (15 July, p. 176)?

Enuresis is one of those bastard problems which are tossed from one physician to another, each hoping that he will not be left with the baby. First we make a vain search for physical cause; then a quick pass to the paediatrician; child-guidance follows with electric gadgets; and finally the psychiatrist. The problem drops back into our laps, so we bless the statistician and say: "He will probably grow out of it by the time he is 10."

The common factor in all the methods of approach mentioned above—and they all have their successes—is suggestion. Opinions on the dangers of symptom removal, although academically valid, make nonsense in light of our daily practice.

The surgeon treats belly-aching by removal of a normal appendix; our gynaecological colleagues cure a multitude of troubles by diagnostic measures alone. In psychiatry is it not possible that we are merely relieving the symptom of depression when we use our modern drugs? We are certainly not curing a disease.

Enuresis is not a hopeless problem. Those who are interested will find that a high rate of success can be achieved in family practice. Patience is essential, along with an established personal relationship with the patient; but record-keeping, conditioning, bribery in the case of the child, suggestion, drugs, and even electric pads will bring success in a majority of cases.—I am, etc.,

Dundee, Angus. R. A. B. RORIE.

Unusual Examples of Adnexal Torsion

SIR,—Torsion of the lateral part of a Fallopian tube after sterilization was first reported by Kohl.[1] Since then other cases have been described.[2–4] It is difficult to say if the part of the tube which twists is healthy before the torsion, or whether it is the seat of a hydrosalpinx. However, the division of the tube deprives the lateral part of any stability which it might obtain from continuity with the medial part, thus facilitating torsion. In this context the following case is of interest.

A 31-year-old prediabetic housewife was Rhesus-negative. Her third baby was affected by maternal antibodies, and survived after an exchange transfusion. The mother was subsequently sterilized in 1963. For nine months before admission she suffered from recurrent abdominal pain, and had been treated for biliary colic, although the biliary tract had not been investigated.

She presented as an emergency with pain and vomiting. Her temperature was 101° F. (38.5° C.). There was tenderness in the right iliac fossa, but no peritonism. A tender cystic mass 3 in. (7.6 cm.) in diameter was found in the right anterolateral fornix. At laparotomy it was noted that a Pomeroy operation had been performed. The lateral part of the right Fallopian tube was cystic and gangrenous, and twisted five times on its mesosalpinx. The biliary tract was healthy. Right salpingo-oophorectomy was performed and the specimen mounted for museum display.

Sterilization by the Pomeroy technique is already under criticism. The pregnancy rate may be 1.7%,[5] while Drake has drawn attention to the occurrence of ectopic pregnancies.[6] Salpingectomy would not guarantee prevention of subsequent pregnancy in or outside the uterus, but would remove the risk of tubal torsion.

A 23-year-old housewife complained of deep dyspareunia after the birth of her first child. The uterus was found to be retroverted, and the ovaries had prolapsed into the pouch of Douglas. The uterus was anteverted without difficulty in the clinic, and a No. 8 Hodge pessary inserted into the vagina. Two hours later the patient developed lower abdominal pain and vomiting. She returned to hospital next day. Her temperature was 98.8°F. (37° C.). There was slight tenderness in the left iliac fossa, but no peritonism. The uterus was still anteverted, but behind it was a tender cystic mass 3 in. (7.6 cm.) in diameter. At laparotomy the left tube and ovary were found to have undergone torsion through one turn and to be gangrenous. The right ovary contained a few small cysts, and lay in the pouch of Douglas. Left salpingo-oophorectomy was performed and the round ligaments shortened. The right ovary was then stitched to the pelvic brim. Microscopy revealed that the left ovary was largely replaced by a cyst 6 in. in diameter.